VELAMENTOUS CORD INSERTION: SIGNIFICANCE OF PRENATAL DETECTION TO PREDICT PERINATAL COMPLICATIONS

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SUMMARY

In the maternal and child health statistics of Japan for 2003, perinatal deaths were most frequent in pregnant women with abnormalities of the placenta, umbilical cord, and fetal membrane. Despite advances in perinatal medicine, approximately 2% of low-risk pregnant women still require an emergency cesarean section after the onset of labor. Because it is likely that half of these cases are associated with placental and umbilical cord abnormalities, it is thought that prenatal detection of such abnormalities would reduce the number of emergency cesarean sections in low-risk women. In our previous studies, some abnormalities of the placenta and umbilical cord were associated with abnormalities of cord insertion. Furthermore, we reported that prenatal detection of velamentous cord insertion (VCI) reduced the number of emergency cesarean sections in low-risk women. In this review, we describe the prenatal detection of abnormalities of umbilical cord insertion and the management of VCI based on our current clinical data. [*Taiwanese J Obstet Gynecol* 2006;45(1): 21–25]

Key Words: velamentous cord insertion, vasa previa, umbilical cord, prenatal diagnosis, ultrasound, placenta previa, variable deceleration

Velamentous Cord Insertion

The umbilical cord normally inserts into the central portion of the placenta, well away from the placental edge. The site of placental cord insertion (CI) is considered abnormal when it is located at the edge of the placental disk (marginal CI) or when the umbilical vessels separate from each other and course between the amnion and chorion before reaching the placenta (velamentous CI, VCI). The rate of VCI ranges from 0.5% to 1.69% of singleton pregnancies [1,2]. It has been reported that abnormal CI is associated with fetal growth restriction, preterm labor, abnormal intrapartum fetal heart rate (FHR) pattern, low Apgar scores at 1 and 5 minutes, neonatal death [1,2], and abruptio placenta [3–5].

In cases with VCI, it is thought that the abnormal FHR pattern and some perinatal complications are caused by lack of Wharton's jelly, which results in compression of vessels during uterine contraction or fetal movement. When the CI site is in the lower uterine segment, VCI and marginal CI are strongly associated with variable decelerations, non-reassuring fetal status, emergency cesarean sections, and other perinatal complications. In our previous report, variable decelerations were observed in 19 of 30 (63%) upper-middle VCIs and four of five (80%) lower VCIs [6,7]. A non-reassuring FHR pattern was observed in four (13.3%) upper-middle VCIs and four (80%) lower VCIs. Emergency cesarean sections were performed in three (10%) upper-middle VCIs and three (60%) lower VCIs. Furthermore, aberrant vessels in lower VCI cases tend to become longer than those in middle and upper VCI (the length of the aberrant vessels was 3.9 ± 3.3 cm in upper-segment

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Received: February 6, 2006
Revised: February 13, 2006
Accepted: February 14, 2006

VCIs, 4.7 ± 4.6 cm in middle-segment VCIs, and 10.6 ± 6.8 cm in lower-segment VCIs), because aberrant vessels are elongated by extension of the uterine isthmus as gestation advances [6,7]. It appears that the lower and longer aberrant vessels of VCI are readily compressed by the fetus, which results in an abnormal FHR pattern and intrapartum complications during labor.

In our previous study, two of 35 VCI cases (6%) and one of 39 marginal CI cases (3%) had placental abruption [6], although the incidence is 0.6–1.0% of all pregnancies [3–5]. Toivonen et al also reported that VCI has a significantly higher risk for placental abruption with an odds ratio of 2.53 [5]. Lower VCI may be associated with extension of the lower uterine segment and atrophy of the chorion villosum that covers the lower segment of the uterus, resulting in abruptio placenta, placenta accreta, accessory placenta, or placental infarction.

In multiple pregnancies, the prevalence of VCI is 10-fold higher than in singleton pregnancies. Only 56.1% of dichorionic diamniotic twins had normal CIs in both placentas. The finding was even more profound for monochorionic diamniotic (MD) twins, of which only 23.8% demonstrated normal CI [8]. Abnormalities of the umbilical cord, particularly VCI, are associated with preterm delivery, twin-to-twin transfusion syndrome (TTTS), and fetal growth restriction [1]. Hanley et al reported a 13-fold increase in the risk of birth weight discordancy in MD twins in the presence of VCI, with a rate of 46% [8]. Fries et al reported that in 38 pairs of MD twins, 11 cases (28.9%) developed TTTS. In these pregnancies, 63.6% had a VCI. The incidences of TTTS in the groups with normal CI and VCI were 18.5% and 58.3%, respectively [9]. Thus, since the influence of VCI is stronger in multiple pregnancies than in single pregnancies, it is thought that prenatal detection of VCI is more important in the management of multiple pregnancies.

Vasa Previa

Vasa previa is a condition in which the umbilical and fetal vessels are unsupported by the placenta or Wharton's jelly and traverse the region of the internal os. Vasa previa is a form of VCI in which the velamentous vessels traverse the fetal membranes in the lower uterine segment. The incidence of vasa previa is estimated at 1:50 VCI cases [10,11] or 1:1,200–5,000 pregnancies [12–15]. Because vasa previa has unsupported fetal vessels below the fetal presenting part, these fetal vessels are easily compressed or ruptured when uterine contractions or membrane rupture occurs, resulting in fetal exsanguination. Since bleeding from vasa previa is of fetal origin, the associated fetal morbidity and mortality are extremely high, ranging from 50% to 60% with intact membranes, and 70% to 100% with ruptured membranes [12,16,17]. An intrapartum clinical diagnosis is rarely made and the diagnosis is very difficult [18].

Catanzarite et al classified vasa previa into two types: Type I is associated with VCI and Type II is associated with vessels that connect between placental lobes and traverse the internal os. The pathophysiology of Type II vasa previa may be explained by atrophy of the partial placenta around the cervical os due to a poor blood supply [18]. They found that of their 10 cases and 23 other reported cases, 54.5% (18/33) were Type II [14,18–23].

Oyelese et al performed a multicenter study of 155 pregnancies complicated by vasa previa [24]. Between 1991 and 2003, 61 of these cases were prenatally diagnosed by ultrasonography/color Doppler and the infant survival rate was 97% (59/61). In cases that were not prenatally diagnosed, the survival rate was 44% (41/94). Multivariable logistic regression analysis revealed that the only significant predictors of neonatal survival were prenatal diagnosis and gestational age at delivery. Thus, high fetal mortality due to vasa previa can be reduced by antenatal diagnosis and elective cesarean section [14,18,24,25].

Several authors have reported vasa previa as well as other placental abnormalities associated with *in vitro* fertilization (IVF) [24,26,27]. Marginal CI and VCI were more frequently found in the IVF population than the general obstetric population. Schachter et al reported that the frequencies of vasa previa and VCI after IVF pregnancy were 1:293 and 1:167, respectively, with odds ratios compared with non-IVF pregnancy calculated as 27.4 (95% confidence interval, CI, 7.4–91.7) and 5.9 (95% CI, 2.5–13.2), respectively. Abnormal placentation and situations that may lead to abnormal placentation are risk factors for vasa previa [27]. Placenta previa and bi-lobed and succenturiate-lobed placentas are risk factors for vasa previa [13,19,23,28], and these are more common in pregnancies achieved after IVF.

Ultrasound Screening

Since pregnancies complicated by VCI, including vasa previa, are at greater risk for adverse perinatal outcome, previous investigators have suggested that systematic ultrasound evaluation of the placental CI site is extremely important [14,25,29,30]. Pretorius et al reported that the detection rate of CI was markedly influenced by

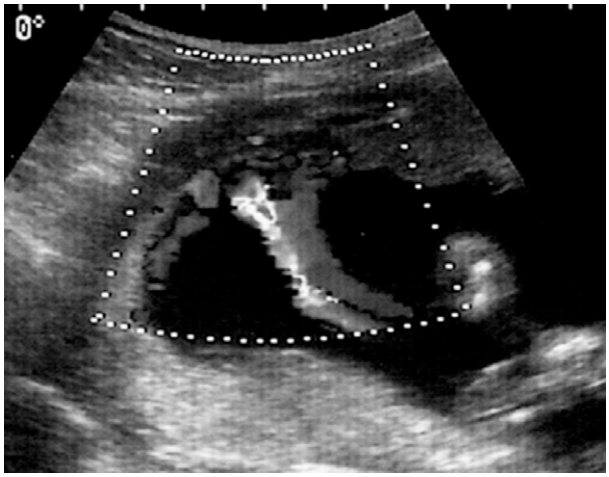

Figure 1. Ultrasound image of velamentous cord insertion in the mid-trimester.

gestational age, ranging from 67% at 15–20 weeks to 30% at 36–40 weeks [30]. As visualization of the placental CI site becomes more difficult with advancing gestation, it should be evaluated in the mid trimester [14,25,31] (Figure 1). One fetal-medicine specialist identified the placental CI site in more than 99% of cases using routine color Doppler scanning in the mid trimester [14,25]. However, in our previous study, antenatal detection of VCI at 18 weeks had a sensitivity of 63% (25/40), specificity of 100% (3406/3406), positive predictive value (PPV) of 100% (25/25), and negative predictive value (NPV) of 99.6% (3406/3421) [6]. Because a number of generalist obstetricians/gynecologists with 3–5 years' experience performed the ultrasound scans in our department, and color Doppler scanning was not used in all cases, the sensitivity of detecting VCI was not high at 18–20 weeks of gestation. Our criteria for ultrasound diagnosis of a VCI were: umbilical vessels entering the placenta margin parallel to the uterine wall and connecting to superficial placental vessels; an immobile CI, even when the uterus is shaken; and umbilical vessels diverging as they traverse the membrane. In fact, the CI site could not be determined more frequently in cases of marginal CI and VCI than in the normal CI. An abnormal CI was present more frequently when it was difficult to image the CI (decreased sensitivity); thus, more precise scanning (in different body positions and using color Doppler) is indicated.

Previous studies have reported that the risk factors for vasa previa include: ultrasound diagnosis of low-lying placenta or placenta previa at earlier gestation; bi-lobed or succenturiate placenta [13,19,23,28]; multiple gestation; suspicion of aberrant vessels [23]; VCI [23,30]; and an IVF pregnancy [24]. Of patients with vasa previa at delivery, 60–69% were diagnosed in the mid-trimester [24,28], whereas, in cases without vasa previa, two of 52 patients (3.8%) were diagnosed as having placenta previa in the mid-trimester [28]. Thus, routine transvaginal color Doppler sonography of the cervical region during the third trimester for women with increased risk is recommended [24,28].

An abnormal CI located in the lower uterine segment is strongly associated with perinatal complications and the pregnancy should be deemed high risk. We think the assessment of CI at first-trimester sonography may improve the sensitivity of detecting CI in the lower uterine segment and have profound implications for management. We reported that in cases in which CI was noted to be in the lower uterine segment during the first trimester, regardless of the location of the placenta at delivery, developmental abnormalities of the placenta and umbilical cord, including low-lying placentas, infarction and abruption of placentas, VCI and marginal CI, occurred frequently [32] (Figure 2). Because of a high visualization rate and short time for detection of cord insertion at 9–11 weeks of gestation, we propose that the systematic identification of CI in the lower uterine segment during the first trimester is extremely useful for identification of high-risk pregnancies, including VCI located in the lower uterine segment and vasa previa. In cases where CI is low at the first-trimester screening, it should be re-evaluated during the third trimester using color Doppler ultrasonography. Through our management of pregnant women, we believe that a safer delivery can be provided with accurate identification of high-risk pregnancies with abnormalities of the placenta and umbilical cord.

Treatment of Pregnancy and Delivery

Variable decelerations and a non-reassuring FHR pattern are frequently observed in cases with VCI. The

Figure 2. Ultrasound image of cord insertion (arrow) into the lower uterine segment at 10 weeks of gestation. In Os = internal os.

increased frequency of variable decelerations is most likely due to the lack of Wharton's jelly in the umbilical vessels, which results in compression of these vessels during uterine contraction or fetal movement. It is thought that variable decelerations occur as a result of cord compression. Lee et al suggest that variation in typical variable deceleration is caused by different degrees of partial cord compression [33]. However, the cause of frequent variable decelerations in VCI is the compression of aberrant vessels and the obstruction of blood flow in both arteries and veins at the same time by the uterine contraction. Variable deceleration without baroreceptor-mediated acceleration (VDNA) frequently occurs in VCI cases (Figure 3). Analysis of VDNA occurrence in the second stage of labor shows that the rate of VDNA is about three times higher in VCI cases than in controls [7].

In cases of VCI and vasa previa, we should offer frequent FHR monitoring in the late third trimester, as long as the pregnancy is not complicated with preterm labor, bleeding, shortening of the cervix, or spontaneous rupture of membranes. Patients should be educated regarding signs and symptoms of preterm of labor. Oyelese et al recommend that women with prenatally diagnosed vasa previa should be offered elective delivery by cesarean section at about 35 weeks of gestation, or earlier if fetal lung maturity is documented [24]. Because we consider that lower VCI is analogous to vasa previa and has a very high rate of non-reassuring fetal status, we recommend elective cesarean section in cases with lower VCI. Other VCI cases can be offered an attempt at vaginal delivery with the backup of an emergency cesarean. During labor, variable deceleration, especially VDNA, should be considered a warning sign of abrupt FHR abnormalities.

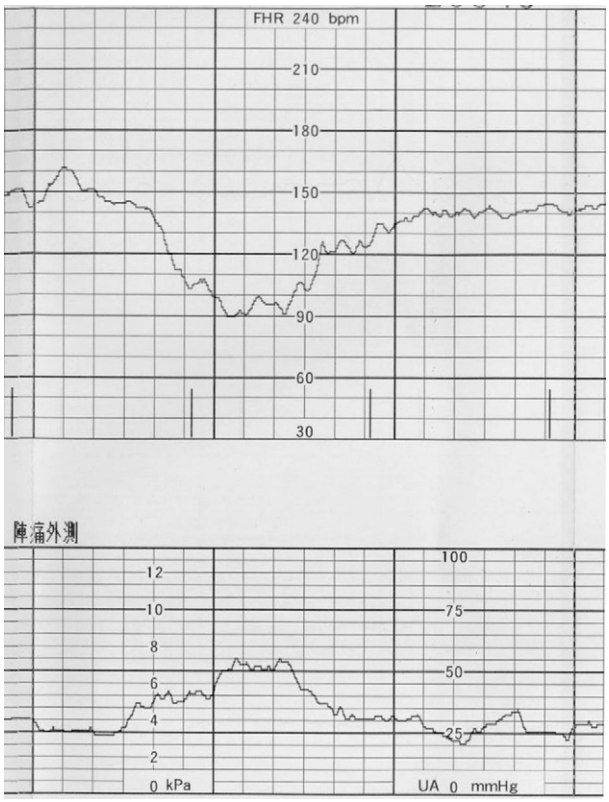

Figure 3. Fetal heart rate pattern named variable deceleration with no acceleration (VDNA).

Conclusion

VCI is associated with increased rates of abnormal FHR tracings and cesarean deliveries. In particular, pregnancies with a VCI located in the lower uterine segment or vasa previa should be deemed high risk. Antenatal diagnosis of a VCI in early gestation improves obstetric management. In cases in which CI is noted to be in the lower uterine segment during the first trimester, developmental abnormalities of the placenta and the umbilical cord (including VCI and vasa previa) occur frequently. Because of a high visualization rate and short time for detection of CI, systematic identification of CI during the first trimester is extremely useful for the identification of high-risk pregnancies.

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